

Serial Peak Expiratory Flow Rates in Patients Undergoing Upper Abdominal Surgeries Under General Anaesthesia and Thoracic Epidural Analgesia

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ABSTRACT

Introduction: Anaesthesia and upper abdominal surgeries alter lung compliance and functional residual capacity resulting from atelectasis. Upper abdominal surgeries also cause a decrease in peak expiratory flow rates, cough reflex due to pain limited inspiration.

Aim: This study aimed to study the effect of thoracic epidural analgesia (TEA) on the peak expiratory flow rates in patients undergoing upper abdominal surgeries.

Materials and Methods: A total of 44 patients posted for elective surgery were enrolled. Group 1 patients received GA + 0.125% bupivacaine infusion TEA and Group 2 received GA + Inj. Diclofenac sodium 50 mg slow i.v. TID for Postoperative analgesia. Haemodynamics, VAS pain score, PEFR measurements were done at 60 minutes, 24 hours, 48 hours and 4 days after surgery in both groups. ABG analysis was taken pre operatively and 24 hours after surgery.

Results: The SBP and DBP values obtained at 60 minutes ($p < 0.016$) 24 and 48 hours ($p < 0.001$) and day 4 ($p < 0.02$)

postoperative showed highly significant difference between the two groups which indicate better haemodynamic parameters in patients receiving epidural analgesia. Postoperatively the difference in PEFR values at 60 minutes, 24 hour, 48 hour and day 4 were very highly significant. ($p < 0.001$). Group1 had a 10.739% deficit on day 4 from its pre operative baseline value while group 2 showed a 34.825 % deficit which was very highly significant ($p < 0.001$). The difference in VAS scores recorded at 60 minutes, 24 hours, 48 hours and day 4 post op were very highly statistically significant ($p < 0.001$). The ABG taken at 24 hours shows statistically significant difference with patients in group 2 showing decreased values in pCO_2 and pO_2 reflecting poorer ventilation and oxygenation.

Conclusion: Thoracic epidural analgesia provides superior analgesia, better cough reflex as seen by better PEFR values, were haemodynamically more stable and their ABG values were better than the NSAID group.

Keywords: Diaphragmatic dysfunction, Hemodynamic stability, Pain relief, PEFR, VAS

INTRODUCTION

Anaesthesia as does surgery alters the ventilatory function beginning with the induction of anaesthesia and often lasting well into the postoperative period. The most frequent problem after upper abdominal and thoracic surgery is atelectasis, which reduces lung compliance and functional residual capacity [1]. Thoracic epidural anaesthesia is used for several surgeries which include upper abdominal, vascular and cardiovascular surgeries. It can contribute to enhanced postoperative outcomes such as improved respiratory function, reduction in ileus and protein sparing due to the adequate pain relief and sympatholysis which allows patients to cough, take deep breaths and mobilize at the earliest [2]. Thoracic Epidural Analgesia/Anaesthesia (TEAA) reduces the incidence of postoperative atelectasis, pneumonia and hypoxaemia [3-7].

Major abdominal and thoracic surgery have a profound effect on the reduction in Functional Residual Capacity (FRC) and this can be attributed to diaphragmatic dysfunction, decreased chest wall compliance and pain during inspiration. The FRC decreases by at least 20% after abdominal surgery, reaching its lowest at 24-48 hours and taking more than one week to return to normal [8]. On the other hand, TEAA with a local anaesthetic and general anaesthesia when compared with intravenous anaesthesia and general anaesthesia resulted in a 27% increase in FRC and an improvement in pulmonary outcome [8]. Pain is said to be a major contributor to the impaired diaphragmatic function and Manikian

and his co-workers in their study found that epidural analgesia partly restored decreased relative contribution of the diaphragm to tidal breathing caused by upper abdominal surgery [9].

Besides TEA, Non-Steroidal anti-Inflammatory Drugs (NSAIDs) have also been used for postoperative analgesia. Joris et al., studied the efficacy of NSAIDs in postoperative pain relief and found that there was a 20 to 50% reduction in opioid consumption, improved quality of analgesia following various types of surgery [10]. Improved respiratory function, sleep quality, and faster recovery of gastrointestinal function have been reported with NSAIDs. However, improved outcome or more rapid recovery with NSAIDs has not been proven [10].

AIM

The aim of this study was to observe the haemodynamic stability postoperatively after TEA, to measure the effect of TEA on PEFR in patients undergoing upper abdominal surgeries and to measure pain scores.

MATERIALS AND METHODS

This study was a randomized controlled trial conducted in tertiary care hospitals attached to Kasturba Medical College, Mangalore over a period of 2 years from November 2009 to October 2011. After obtaining approval from Institutional Human Ethics Committee, an informed written consent was obtained from all patients. The study was performed on American society of Anaesthesiologists

(ASA) physical status 1 and 2 with an age range of 20-55 years scheduled for upper abdominal surgeries. Patients were assigned to one of the two groups.

Group I: General Anaesthesia + postoperative Thoracic Epidural Analgesia (TEA).

Group II: General Anaesthesia + postoperative NSAIDs for analgesia.

The surgeries included Open cholecystectomies and Gastric surgeries. Any patient who refused, had history of allergy to local anaesthetics, had a low platelet count (<1,00,000), had abnormal coagulation profile, bony spinal deformities, neurological disorders, sepsis or infection at the puncture site of epidural were excluded from the study.

Detailed preoperative assessment including history followed by relevant investigations which included Complete Blood Count (CBC), coagulation profile, serum biochemistry, Arterial Blood Gas (ABG) analysis, Electrocardiography (ECG), Chest Radiography (CXR). Every patient was assessed for epidural catheter placement. They were taught the technique of pulmonary peak flow rate measurement using a peak flow meter (PEFR). They were explained how to use the Visual Analogue Scale (VAS).

The PEFR test was performed using a peak expiratory flow monitor. The patient was asked to breathe in as deeply as possible and then blow into the instrument's mouthpiece as hard and fast as possible. Measurements were made with the patient in supine position with a pillow under the head. The highest value of three measurements was recorded.

On arrival in Operation Theatre (OT), appropriate gauge Intravenous (IV) cannulas, baseline monitors such as electrocardiography, non-invasive blood pressure monitoring, pulse oximetry were used. Post induction, end tidal capnography and temperature were monitored. All the cases were conducted under general anaesthesia. Premedication was given with Injection fentanyl 2mcg/kg 5 minutes before induction. After pre oxygenation for 3-5 minutes; patient was induced with injection propofol titrated dose till loss of verbal response. Neuromuscular blockade was given with injection vecuronium 0.1mg/kg. Patient was intubated with appropriate size oral cuffed ET tube after 3 minutes. Anaesthesia was maintained with controlled mechanical ventilation using closed circuit with $O_2:N_2O :: 1:1$ ratio and Isoflurane at 1%. Relaxation was maintained with injection vecuronium bromide 1 mg top ups IV. Intraoperatively analgesia was maintained with boluses of Inj. Fentanyl 50 mcg boluses if there were changes in haemodynamics suspected to be due to pain. At the end of surgery, neuromuscular blockade was antagonized with Inj. Neostigmine 0.05 mg/kg and Inj. Glycopyrrolate 0.01 mg/kg.

Group 1: 18 G epidural catheter was inserted by "Loss of resistance" technique at the lower thoracic vertebral level 20 minutes before induction. A 3 ml of lignocaine adrenaline test dose was given. This was started as an infusion of 0.125% at the rate of 5 ml/h for all patients in this group after patient was extubated.

Group 2: Systemic NSAIDs, Injection Diclofenac sodium 50 mg slow i.v. TID was given for analgesia postoperatively in these patients.

Postoperatively, VAS pain score, PEFR measurements were done at 60 minutes, 24 hours, 48 hours and 4 days after surgery in both the groups. A preoperative and postoperative arterial blood gas sample was taken for analysis at 24 hours.

STATISTICAL ANALYSIS

Data obtained from the study was analysed using the computer software Statistical Package for Social Sciences (SPSS) version 10. Data are expressed in Frequency, Percentage, Mean and \pm Standard deviation. To elucidate the associations and comparisons between different parameters, Chi-Square Test was used as the

non parametric test. Student's t-test was used to compare the mean value between 2 groups. To compare different groups with each other, non parametric Mann-Whitney's U test was employed. For all statistical evaluations, a two tailed probability p-value of <0.05 was considered significant.

RESULTS

To minimize any potential risk to the subjects, the present study involved ASA grade physical status 1 and 2 patients, thus the quantitative data of this study are only applicable to such patients. From [Table/Fig-1,2] it is observed that patients in both groups were comparable with regard to gender, age, height and weight as no significant difference was observed between the two groups. All patients showed changes both in haemodynamics (pulse rate and blood pressure), pain scores as well as PEFR (Peak Expiratory Flow Rate). The pulse rate was lesser in the epidural group (Group 1) at 60 mins, 24 hour and 48 hour postoperative day with values of 67.90 ± 6.33 , 73 ± 6.21 and 73 ± 4.03 respectively as compared to 78.30 ± 9.44 , 82.86 ± 8.69 and 79.17 ± 5.85 of the NSAID group (Group 2) as seen in [Table/Fig-3]. The mean blood pressure values were statistically very highly significant in group 1 at 24 hour (91.23 ± 6.34), 48 hour (91.61 ± 6.48) and 4 days (91.74 ± 7.003) as compared to group 2 at 24 hour ($104.72.56 \pm 8.66$), 48h (98.28

Number of patients	Group		Total
	Group 1	Group 2	
Male	15(71.4%)	14(60.9%)	29(65.9%)
Female	6(28.6%)	9(39.1%)	15(34.1%)
Total	21(100%)	23(100%)	44(100%)

[Table/Fig-1]: Gender distribution of study participants.

$\chi^2=0.545$ p=0.46

Patient Profile	Group 1 N = 21	Group 2 N = 23	p-value
Age	45 ± 7.071	45.48 ± 5.517	0.803
Weight (kgs)	58.67 ± 9.134	58.09 ± 6.494	0.808
Height (cms)	167.9 ± 9.099	166.35 ± 8.973	0.575

[Table/Fig-2]: Demography.

Data presented as mean \pm SD

Pulse rate (beats /min)	Group 1 (n=21)	Group 2 (n=23)	p-value
pre op	73.53 ± 4.19	72.43 ± 4.74	0.426
60 min postop	67.90 ± 6.33	78.30 ± 9.44	<0.001
24 hrs postop	73.04 ± 6.21	82.86 ± 8.69	<0.001
48 hrs post op	73.04 ± 4.03	79.17 ± 5.85	<0.001
4 days postop	72.76 ± 4.21	78.13 ± 5.49	0.001

[Table/Fig-3]: Pulse rate.

Data presented as mean \pm SD

Characteristics		Group 1 (n=21)	Group 2 (n=23)	p-value
Blood pressure (mm of Hg)	Pre op	92.82 ± 9.36	89.01 ± 9.66	0.192
	60 mins post op	83.74 ± 9.52	93.01 ± 14.38	0.015
	24 hrs post op	91.23 ± 6.34	$104.72.56 \pm 8.66$	<0.001
	48 hrs post op	91.61 ± 6.48	98.28 ± 7.09	0.002
	4 days post op	91.74 ± 7.003	96.05 ± 7.61	0.058

[Table/Fig-4]: Mean Blood pressure.

Data are presented as mean \pm SD.e.

PEFR (L/min)	Group 1 (n=21)	Group 2 (n=23)	p-value
Pre op	408.57 ± 81.99	454.78 ± 82.28	0.069
60 mins post op	310.95 ± 68.76	254.78 ± 59.30	0.006
24 hrs post op	319.04 ± 71.19	253.47 ± 55.97	< 0.001
48 hrs post op	351.42 ± 75.64	281.30 ± 51.01	< 0.001
Day 4 post op	363.80 ± 72.42	293.04 ± 44.56	< 0.001

[Table/Fig-5]: Peak expiratory flow rates.

Data presented as mean \pm SD

	Group 1 (n=21)	Group 2 (n=23)	p-value
Percentage difference (%)	10.739 ± 5.04	34.82 ± 6.77	< 0.001

[Table/Fig-6]: Percentage difference of PEFR between the study groups from pre op vs Day 4.
Data presented as mean ± SD

VAS	Group1 (n=21)	Group 2 (n=23)	p- value
pre op	1.85±0.79	1.34±0.71	0.03
60mins post op	1.71±0.56	3.43±1.50	<0.001
24 hrs post op	2.09±1.04	4.39±1.33	<0.001
48 hrs post op	1.09±0.30	3.30±1.33	<0.001
Day 4 post op	1.04±0.21	2.78±0.73	<0.001

[Table/Fig-7]: Visual analogue score.
Data presented as mean ± SD

		Group1 (n=21)	Group 2 (n=23)	p- value
pH	pre op	7.39 ± 0.03	7.38 ± 0.03	0.39
	post op	7.42 ± 0.03	7.37 ± 0.09	0.013
pCo2	pre op	35.72 ± 2.95	34.42 ± 4.05	0.259
	post op	38.41 ± 3.80	35.76 ± 1.35	0.004
pO2	pre op	100.46 ± 14.38	96.59 ± 8.76	0.287
	post op	90.42 ± 9.12	82.56 ± 3.94	0.001
BE	pre op	-1.01 ± 1.96	-1.18 ± 1.57	0.761
	post op	-0.40 ± 1.14	0.90 ± 1.84	0.008

[Table/Fig-8]: ABG Analysis.

Data presented as mean ± SD
pCo2= partial pressure of carbon dioxide, pO2 = partial pressure of oxygen, BE= base excess

± 7.09) and 4 days (96.05 ± 7.61) postoperatively reflected by value in [Table/Fig-4]. In this study, as shown in [Table/Fig-5], peak expiratory flow rate preoperatively was not significant in both groups ~425L/min. However, the decrease in Peak Expiratory Flow Rate (PEFR) was 21.92% and 14% in group 1 at 24 hours and 48 hours postoperatively, while the decrease was 44.27% and 38.15% in group 2 at 24 hours and 48 hours postoperatively. The lowest PEFR values were observed at 60 minutes and 24 hours post procedure, more significant in group 2 as compared to group 1. The average decrease from baseline was -90 litre/min in group 1 and -200 litre/min in group 2, thereby showing highly significant reduction in PEFR values.

In the present study it was also observed that even though the PEFR values did not reach baseline preoperative values, the thoracic epidural group showed only a 10.739 ± 5.04% deficit as compared to a 34.82±6.77% deficit in the NSAIDs group on day 4 postoperatively as shown in [Table/Fig-6].

[Table/Fig-7] shows, the VAS scores recorded during the study. VAS scores were observed to be higher in group 2 at 60 minutes (3.43± 1.50), 24 hours (4.39± 1.33), 48 hours (3.30±1.33) and 4 days (2.78± 0.73) after surgery as compared to group 1 who had minimal pain score values at 60 minutes (1.71 ± 0.56), 24 hours (2.09±1.04), 48 hours (1.09 ±0.3) and 4 days (1.04 ± 0.21). Highest pain recorded using VAS was observed at 24 hours postoperatively and more significantly in group 2. This corresponds to the pain limited effort in breathing which also reflected low PEFR values. From the data in [Table/Fig-8], the ABG analysis shows, no significant difference between the two groups preoperatively while the ABG taken at 24 hours shows, statistically significant difference with respect to the pH, PCO₂, PO₂ and base excess. Group 2 showed lower PO₂ and PCO₂ values compared to group 1 indicating decreased oxygenation and hyperventilation respectively.

DISCUSSION

The effects of abdominal surgery and general anaesthesia on pulmonary function have been extensively studied. Pain is

an important contributor to inspiratory muscle dysfunction after upper abdominal surgery and TEA may partly reverse this dysfunction. Respiratory muscle dysfunction is well documented after upper abdominal surgery [11,12]. The cause for the dysfunction has been said to be due to irritation, inflammation, and trauma in the vicinity of the diaphragm, leading to local mechanical failure [12]. GT Ford et al., studied 15 patients undergoing upper abdominal surgeries and his data indicate reduced diaphragm activity in the postoperative period, with a shift from predominantly abdominal to rib cage breathing [13]. There was a reversal toward normal function by 24 hour. This reduction in diaphragm function may be responsible for the atelectasis, reduced vital capacity, and hypoxaemia in postoperative patients. This was confirmed in the present study, in which PEFR was found to be significantly reduced at 60 minutes, 24 hour and 48 hour after upper abdominal surgery and from the ABG analysis. The nature of the postoperative muscle dysfunction remains elusive. The findings in this study are also in agreement with the findings of Boysen et al., who studied respiratory functions after upper abdominal surgeries and concluded that lung volumes are reduced to their least at 24 to 48 hours [14]. The study conducted by Wahba et al., showed similar results with FRC and VC [8]. The FRC and VC values of 84% and 55% postoperatively were observed with the use of epidural analgesia as compared to the control group which showed 78% and 38% having received narcotic analgesic drugs postoperatively. In this study, TEA was associated with a significant improvement in pulmonary function during the first four postoperative days as compared to the other group, using PEFR as a surrogate for pulmonary function.

Sympathetic activation associated with surgery and postoperative pain manifests as tachycardia, hypertension and increased contractility, all of which serve to increase myocardial oxygen consumption. Vera et al., in his study of the preferred anaesthetic technique for thoracic surgery concluded that patients may benefit from general anaesthesia and thoracic epidural anaesthesia with respect to oxygenation and haemodynamic stability [15]. Blomberg S et al., studied the effects of high TEA on central haemodynamics as measured by pulmonary arterial catheterization in nine patients with severe coronary artery disease and unstable angina pectoris [16]. During ischemic chest pain, pulmonary artery and pulmonary capillary wedge pressures are significantly increased. TEA, while relieving the chest pain, significantly decreased systolic arterial blood pressure, heart rate, and pulmonary artery and pulmonary capillary wedge pressures, without any significant changes in coronary perfusion pressure, cardiac output, stroke volume and systemic or pulmonary vascular resistances.

LIMITATION

- The study population was from a limited number of academic medical centers with corresponding geographic and demographic diversity.
- The pain scores may have been underreported as they relied mainly on patient self-reporting.
- The sample size of the study was fairly small.

CONCLUSION

As per this study results, TEA provides superior analgesia and a better cough reflex. The PEFR values were good in the study population and they were haemodynamically more stable.

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